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=> s prostaglandin (w)synthase and fluorescence (w) polarization
L1 5 PROSTAGLANDIN (W) SYNTHASE AND FLUORESCENCE (W) POLARIZATION

=> d ibib abs l1 1-5

L1 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:162567 CAPLUS
DOCUMENT NUMBER: 140:212973
TITLE: **Fluorescence polarization** assay for screening **prostaglandin synthase** inhibitors
INVENTOR(S): Ma, Y. Henry; Li, Zhuyin; Xiong, Junjie; Sabol, Jeffrey S.
PATENT ASSIGNEE(S): Aventis Pharmaceuticals Inc., USA; Hu, Linghong
SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004016223	A2	20040226	WO 2003-US25766	20030815
WO 2004016223	A3	20041021		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2495391	AA	20040226	CA 2003-2495391	20030815
US 2004082021	A1	20040429	US 2003-642468	20030815
EP 1543327	A2	20050622	EP 2003-788590	20030815

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

PRIORITY APPLN. INFO.: US 2002-404008P P 20020816
GB 2002-29244 A 20021216
WO 2003-US25766 W 20030815

AB Provided herein is a novel and useful method for evaluating the ability of compds. or agents to decrease the activity of microsomal prostaglandin E synthase (mPGES) or hematopoietic prostaglandin D synthase (hPGDS) to produce their resp. prostaglandin products. Such a method of the present invention comprises the steps of mixing the **prostaglandin synthase** with its substrate, a cofactor and the compound or agent so that the enzymic reaction can occur. The mixture is then incubated with a stop solution comprising an agent that prevents the spontaneous conversion of unreacted substrate into the prostaglandin product. This mixture is then incubated with a detection reagent that comprises the prostaglandin product labeled with a fluorescent label (i.e. a tracer), and an antibody having the prostaglandin product as an immunogen. Subsequently, the mixture and a control mixture that has been treated in the identical fashion, but lacks the compound or agent, are illuminated with plane polarized light having a wavelength at which the fluorescent label fluoresces. The **fluorescence polarization** of the mixture and the control mixture are measured and compared. A mixture having a polarization measurement greater than the polarization measurement of the control mixture indicates that the compound or agent decreased the activity of the **prostaglandin synthase**. The present invention is based upon the discovery that surprisingly and unexpectedly, **fluorescence polarization** can be used to identify compds. or agents that decrease the activity of a **prostaglandin synthase**, e.g., mPGES or hPGDS, to produce a prostaglandin, e.g., PGD2 or PGE2. An assay has been developed to measure the conversion of PGH2 to PGE2 by inducible microsomal PGE2 synthase. The assay is configured based on the **Fluorescence Polarization** principle. The enzyme is incubated with PGH2, glutathione, and the compound or agent being evaluated. After a short incubation period (at least 30 s), a stop solution containing FeCl2 and citric acid is added to quench any remaining PGH2, which would otherwise undergo spontaneous conversion to PGD2 or PGE2, and thus interfere with the quantification of the enzymic conversion of PGH2 to PGE2. A detection solution containing a fluorescence labeled (Texas Red) tracer (PGE2) and anti-PGE2 antibody is then added in order to generate the specific signal that is inversely proportional to the production of PGE2. The PGE2 generated from the enzymic reaction will compete specifically for the antibody and release the fluorescence labeled tracer. Inhibition of PGE2 synthase activity will result in increased FP value. The assay described above was validated with a known inhibitor of mPGES. A **fluorescence polarization** (FP) assay to measure hPGDS activity has also been developed. The assay described above was validated using HQL 79, a known inhibitor of hPGDS.

L1 ANSWER 2 OF 5 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1984:14141306 BIOTECHNO
TITLE: Inhibition of platelet function by cis-unsaturated fatty acids
AUTHOR: Euan MacIntyre D.; Hoover R.L.; Smith M.; et al.
CORPORATE SOURCE: Department of Surgery, Beth Israel Hospital, Boston, MA 02215, United States.
SOURCE: Blood, (1984), 63/4 (848-857)
CODEN: BLOOAW
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English

AN 1984:14141306 BIOTECHNO

AB The uptake of free fatty acids has previously been shown to affect the capping of lymphocytes, and there is evidence that different types of fatty acids may partition into separate lipid domains in cell surface

membranes. In studies of gel-filtered human platelets, we found that cis-unsaturated fatty acids (1-35 μM) inhibited platelet shape change, aggregation, and secretion of 5-hydroxytryptamine induced by thrombin, adenosine disphosphate (ADP), collagen, U46619 (a thromboxane A.sub.2 analog), or plant lectins, but not that induced by A23187, a calcium ionophore. Trans-unsaturated and saturated fatty acids had little or no inhibitory effect. The inhibitory effects of cis-unsaturated fatty acids were not affected by inhibition of adenylate cyclase or cyclooxygenase. .sup.1.sup.4C-labeled fatty acids were taken up into platelet lipids. The maximum platelet inhibitory effect of cis-unsaturated fatty acids were seen when over 90% of the platelet label was still in the form of free fatty acids. Platelet inhibition could be reversed by washing the platelets by gel filtration. Binding of platelet agonists to the platelet was not inhibited by the fatty acids. Cis-unsaturated fatty acids, but not trans-unsaturated or saturated fatty acids, decreased **fluorescence polarization** of platelets or isolated platelet membranes monitored with 1,6-diphenyl-1,3,5-hexatriene. The potency of the fatty acids as inhibitors of platelet aggregation was inversely correlated with their melting points. These data suggest that inhibition of receptor-mediated platelet responses by cis-unsaturated fatty acids results from perturbation of the platelet membrane in specific lipid domains.

L1 ANSWER 3 OF 5 EMBASE

ACCESSION NUMBER: 2000080358 EMBASE

TITLE: Modulation of LH/hCG receptors and physical state of ovarian membranes in rat pseudopregnancy.

AUTHOR: Jezova M.; Sczukova S.; Vranova J.; Kolena J.

CORPORATE SOURCE: M. Jezova, Inst. of Experimental Endocrinology, Slovak Academy of Sciences, Vlarska 3, 833 06 Bratislava, Slovakia. ueenjez@savba.savba.sk

SOURCE: General Physiology and Biophysics, (1999) Vol. 18, No. 4, pp. 347-356. .
Refs: 31
ISSN: 0231-5882 CODEN: GPBIE2

COUNTRY: Slovakia

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 003 Endocrinology
010 Obstetrics and Gynecology
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20000316
Last Updated on STN: 20000316

AB Previous investigations have demonstrated that increased ovarian function during pseudopregnancy in the rat may be associated with alterations of the physical state of membranes. Changes in rigidity of membrane lipids were observed during the formation as well as regression of corpora lutea. The effects of cyclooxygenase inhibitors (indomethacin and acetylsalicylic acid (ASA)) and of selected steroids (estradiol, testosterone and dihydrotestosterone) on the functional state of luteinized ovaries were studied. The compounds were administered to the animals in silastic capsules on different days after hCG injections. ASA and indomethacin administration on days 10 and 11 after hCG injection resulted in an increase in the LH/hCG receptor binding activity and rigidity of ovarian membrane lipids, as determined by **fluorescence polarization** of 1,6-diphenyl-1,3,5 hexatriene (DPH) probe. This effect was apparent within 7 days after indomethacin and ASA treatment. Both estradiol and testosterone significantly increased the ovarian LH/hCG binding activity, however estradiol did not affect the membrane lipid rigidity. Unlike testosterone, the administration of dihydrotestosterone induced a decrease in membrane lipid rigidity and reduced the accessibility of the LH/hCG receptor. Inhibitors of prostaglandin

F(2α) (PGF(2α)) synthesis, as the endogenous mediator of luteolysis, were shown to delay the regression of the corpora lutea and to prolong the luteal activity in pseudopregnant rats.

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ACCESSION NUMBER: 92336569 EMBASE
DOCUMENT NUMBER: 1992336569
TITLE: Decreased platelet membrane fluidity due to glycation or acetylation of membrane proteins.
AUTHOR: Winocour P.D.; Watala C.; Perry D.W.; Kinlough-Rathbone R.L.
CORPORATE SOURCE: Department of Pathology, McMaster University, 1200 Main Street West,Hamilton, Ont. L8N 3Z5, Canada
SOURCE: Thrombosis and Haemostasis, (1992) Vol. 68, No. 5, pp. 577-582. .
ISSN: 0340-6245 CODEN: THHADQ
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
025 Hematology
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 921213
Last Updated on STN: 921213

AB Platelets from diabetic subjects and animals are hypersensitive to agonists in vitro. Membrane fluidity modulates cell function and previously we observed reduced membrane fluidity in platelets from diabetic patients associated with hypersensitivity to thrombin. We previously reported that decreased fluidity of isolated platelet membranes from diabetic patients is associated with increased glycation of platelet membrane proteins, but not with any change in the cholesterol to phospholipid molar ratio. We have now examined in vitro whether incubation of platelet membranes in a high glucose medium causes sufficient glycation to reduce membrane fluidity. Incubation of platelet membranes from control subjects in a high glucose (16.1 mM) medium for 10 days at 37°C led to an increase in the extent of glycation of membrane proteins and a decrease in membrane fluidity (indicated by an increase in steady state **fluorescence polarization**); most of the changes occurred within the first 3 days of incubation. Incubation of platelet membranes with 5.4 mM glucose had less effect. In contrast, incubation of platelet membranes with the same concentrations of 1-0-methylglucose did not cause a change in either the extent of glycation of proteins or membrane fluidity. We also determined if acetylation by aspirin or acetyl chloride of the sites available for glycation on platelet membrane proteins leads to a similar reduction in membrane fluidity. Pretreatment of platelet membranes with aspirin or acetyl chloride diminished the extent of glycation that occurred when platelet membranes were subsequently incubated with glucose, but membrane fluidity was reduced even in the absence of glucose: subsequent incubation with glucose caused no further reduction in membrane fluidity. Similar results were obtained when red blood cells were incubated with high concentrations of glucose or methyl glucose either with or without pretreatment with aspirin or acetyl chloride. Further experiments using platelet membranes showed that the reduction in membrane fluidity due to aspirin was independent of its acetylating effect on platelet cyclo-oxygenase. Ingestion of aspirin also caused a reduction in membrane fluidity of platelets. Therefore, glycation of platelet membrane proteins reduces membrane fluidity, but the effect results from occupation of the sites available for glycation and not the presence of glucose moieties per se at these sites. Acetylation of platelet membrane proteins either in vitro or

in vivo also reduces membrane fluidity; this effect is not associated with platelet hypersensitivity to thrombin.

L1 ANSWER 5 OF 5 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 84116254 EMBASE
DOCUMENT NUMBER: 1984116254
TITLE: Inhibition of platelet function by cis-unsaturated fatty acids.
AUTHOR: Euan MacIntyre D.; Hoover R.L.; Smith M.; et al.
CORPORATE SOURCE: Department of Surgery, Beth Israel Hospital, Boston, MA 02215, United States
SOURCE: Blood, (1984) Vol. 63, No. 4, pp. 848-857. .
CODEN: BLOOAW
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
006 Internal Medicine
033 Orthopedic Surgery
LANGUAGE: English
ENTRY DATE: Entered STN: 911210
Last Updated on STN: 911210

AB The uptake of free fatty acids has previously been shown to affect the capping of lymphocytes, and there is evidence that different types of fatty acids may partition into separate lipid domains in cell surface membranes. In studies of gel-filtered human platelets, we found that cis-unsaturated fatty acids (1-35 μM) inhibited platelet shape change, aggregation, and secretion of 5-hydroxytryptamine induced by thrombin, adenosine disphosphate (ADP), collagen, U46619 (a thromboxane A2 analog), or plant lectins, but not that induced by A23187, a calcium ionophore. Trans-unsaturated and saturated fatty acids had little or no inhibitory effect. The inhibitory effects of cis-unsaturated fatty acids were not affected by inhibition of adenylate cyclase or cyclooxygenase. 14C-labeled fatty acids were taken up into platelet lipids. The maximum platelet inhibitory effect of cis-unsaturated fatty acids were seen when over 90% of the platelet label was still in the form of free fatty acids. Platelet inhibition could be reversed by washing the platelets by gel filtration. Binding of platelet agonists to the platelet was not inhibited by the fatty acids. Cis-unsaturated fatty acids, but not trans-unsaturated or saturated fatty acids, decreased **fluorescence polarization** of platelets or isolated platelet membranes monitored with 1,6-diphenyl-1,3,5-hexatriene. The potency of the fatty acids as inhibitors of platelet aggregation was inversely correlated with their melting points. These data suggest that inhibition of receptor-mediated platelet responses by cis-unsaturated fatty acids results from perturbation of the platelet membrane in specific lipid domains.

=> s fluorescence and Texas (w) red and prostaglandin
L2 1 FLUORESCENCE AND TEXAS (W) RED AND PROSTAGLANDIN

=> s fluorescence and prstaglandin and antibody and label
L3 0 FLUORESCENCE AND PRSTAGLANDIN AND ANTIBODY AND LABEL

=> s fluorescence and prostraglandin and antibody and label
L4 0 FLUORESCENCE AND PROSTRAGLANDIN AND ANTIBODY AND LABEL

=> s fluorescence and prostaglandin and antibody and label
L5 6 FLUORESCENCE AND PROSTAGLANDIN AND ANTIBODY AND LABEL

=> d ibib l2 1

L2 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:162567 CAPLUS

DOCUMENT NUMBER: 140:212973
TITLE: **Fluorescence** polarization assay for screening **prostaglandin** synthase inhibitors
INVENTOR(S): Ma, Y. Henry; Li, Zhuyin; Xiong, Junjie; Sabol, Jeffrey S.
PATENT ASSIGNEE(S): Aventis Pharmaceuticals Inc., USA; Hu, Linghong
SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004016223	A2	20040226	WO 2003-US25766	20030815
WO 2004016223	A3	20041021		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2495391	AA	20040226	CA 2003-2495391	20030815
US 2004082021	A1	20040429	US 2003-642468	20030815
EP 1543327	A2	20050622	EP 2003-788590	20030815
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				

PRIORITY APPLN. INFO.:

US 2002-404008P	P	20020816
GB 2002-29244	A	20021216
WO 2003-US25766	W	20030815

=> d ibib abs l5 1-6

L5 ANSWER 1 OF 6 MEDLINE on STN
ACCESSION NUMBER: 1999366819 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10440207
TITLE: Intraportal perfusion of **prostaglandin** E1 attenuates hepatic postischaemic microcirculatory impairments in rats.
AUTHOR: Iwata K; Shimazu M; Wakabayashi G; Ohshima A; Yoshida M; Kitajima M
CORPORATE SOURCE: Department of Surgery, Keio University School of Medicine, Tokyo, Japan.
SOURCE: Journal of gastroenterology and hepatology, (1999 Jul) 14 (7) 634-41.
Journal code: 8607909. ISSN: 0815-9319.
PUB. COUNTRY: Australia
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 19991014
Last Updated on STN: 19991014
Entered Medline: 19991004
AB BACKGROUND: The efficacy of intraportal perfusion with **prostaglandin** E1(PGE1) in decreasing postischaemic hepatic microcirculatory damage was studied in rats. METHODS: An extrahepatic portosystemic shunt was created by attaching the spleen to a subcutaneous

site on the left lateral wall of the abdomen in male Wistar rats weighing between 200 and 350 g. Four weeks later, when the shunt was mature, the portal vein and hepatic artery were occluded for 60 min. The animals were divided into the following three groups according to the type of intraportal perfusion during the ischaemic phase: group 1 consisted of untreated animals; group 2, animals perfused with lactated Ringer's solution; and group 3, animals perfused with PGE1 (0.1 microg/kg per min). The hepatic microcirculation was observed under an inverted intravital microscope after the injection of fluorescent dyes to **label** leucocytes and damaged cells 30 and 60 min after reperfusion. The liver was removed 60 min after reperfusion and stained immunohistochemically using 1A29, an anti-rat intercellular adhesion molecule-1 (ICAM-1) **antibody**. RESULTS: The leucocyte velocity during reperfusion was lowest in group 1 and highest in group 3. Of the three groups, group 3 showed the least leucocyte adhesion to the sinusoidal walls and terminal venules, the lowest damaged cell count and the lowest ICAM-1 expression on the sinusoidal walls. CONCLUSION: The results of this study suggest that hepatic perfusion with PGE1 markedly alleviates microcirculatory damage associated with ischaemia and reperfusion through the inhibition of leucocyte-endothelium interactions.

L5 ANSWER 2 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:198856 BIOSIS
DOCUMENT NUMBER: PREV200200198856
TITLE: The gC1qR/p33 is constitutively expressed on the surface membrane of human platelets.
AUTHOR(S): Peerschke, Ellinor I. [Reprint author]; Murphy, Tara K. [Reprint author]; Ghebrehiwet, Berhane
CORPORATE SOURCE: Pathology, Weill Medical College of Cornell University, New York, NY, USA
SOURCE: Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 516a. print.
Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 1. Orlando, Florida, USA. December 07-11, 2001. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Mar 2002
Last Updated on STN: 20 Mar 2002

AB The gC1qR is an acidic cellular glycoprotein, which was originally described as a binding protein for the globular domain of the complement subcomponent C1q. Additional studies have shown that gC1qR interacts with a variety of cellular, viral, bacterial, and plasma proteins, and is expressed by many somatic and cultured cells including platelets, and endothelial cells, but not red blood cells (RBC). It's interaction with prothrombin, thrombin, and high molecular weight kininogen suggests a potential role for gC1qR in thrombosis and inflammation. The observation that transfected cell lines expressed recombinant gC1qR predominantly in mitochondria, coupled with the inability of many investigators to surface **label** cells with anti-gC1qR **antibodies**, has led to considerable controversy with regard to the surface membrane expression of this multiligand binding protein and its biologic function. We now present evidence demonstrating the surface membrane expression of gC1qR on human blood platelets. Cell surface gC1qR was examined by **fluorescence** activated cell sorting (FACS), comparing the reactivity of two monoclonal **antibodies**, 74.5.2, recognizing gC1qR carboxyterminal amino acids 204-218, and 60.11, directed against the gC1qR amino terminal amino acids 76-93. Anti-gC1qR **antibody** binding was detected using an Alexa-488 conjugated goat-anti mouse secondary **antibody**. In a typical experiment, 74.5.2 labeled apprx90% of washed platelet in 0.01 M HEPES-buffered modified Tyrode's

solution (pH 7.5) with a mean peak **fluorescence** (MPF) of apprx300 units. In comparison, control, nonimmune mouse IgG labeled apprx15% of platelets (MPF 11). Platelet surface labeling with 74.5.2 more than doubled in the presence of 50 muM ZnCl2 (MPF 709). This zinc-dependent increase in 74.5.2 binding was concentration dependent and approached a maximum at 50muM ZnCl2. When the 60.11 monoclonal **antibody** was used for platelet membrane gC1qR detection, however, only apprx20% of platelets became labeled (MPF 48). The proportion of labeled platelets and the **fluorescence** intensity increased in the presence of zinc, with apprx63% of platelets binding 60.11 (MPF 85) in the presence of 50muM ZnCl2. The **fluorescence** intensity of platelet labeling with 60.11, however, never approached that observed with 74.5.2, even when higher concentrations of 60.11 were used. No increase in gC1qR expression was noted following platelet activation with either the combination of ADP (20muM) pins epinephrine (10 muM) or TRAP (10muM). Furthermore, preincubation of platelets with 0.1 muM PGE1 had no effect on anti-gC1qR **antibody** binding. RBC, which do not possess detectable gC1qR by Western blotting, failed to react with either 74.5.2 or 60.11 in the presence or absence of zinc. Taken together, these studies provide definitive evidence for the constitutive, surface membrane expression of gC1qR on human blood platelets, and suggest that monoclonal **antibodies** 74.5.2 and 60.11 recognize a zinc-dependent conformation of gC1qR. Moreover, these data demonstrate that detection of gC1qR on cell surfaces may be highly dependent on **antibody** specificity. Finally, since the binding of high molecular weight kininogen to gC1qR was shown previously to be zinc-dependent, results from the present study support the concept that zinc ions may be involved in the functional modulation of gC1qR on the platelet surface.

L5 ANSWER 3 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1990:243340 BIOSIS
DOCUMENT NUMBER: PREV199089130293; BA89:130293
TITLE: CYCLOPHOSPHAMIDE AND 15S-15 METHYL PGE-1 CORRECT THE T-B LYMPHOCYTE RATIOS OF NZB-NZW MICE.
AUTHOR(S): GIRARD D [Reprint author]; ALOISI R M; BLIVEN M L; CUNNINGHAM A C; OTTERNESS I G
CORPORATE SOURCE: CENT RES DIV, PFIZER INC, GROTON, CONN 06340, USA
SOURCE: Agents and Actions, (1990) Vol. 29, No. 3-4, pp. 333-341. CODEN: AGACBH. ISSN: 0065-4299.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 19 May 1990
Last Updated on STN: 19 May 1990

AB The lupus of NZB/NZW F1 female mice is associated with immune complex glomerulonephritis and premature death. Cyclophosphamide and 15(S)-15 methyl PGE1 therapy halt disease progression. Fluorescein conjugated **antibodies** were utilized to **label** specfic leukocytes and the subsets were quantitated using a **Fluorescence** Activated Cell Sorter. Normal outbred CD-1 female mice showed a decrease in absolute T and B cell numnbers with age, but the ratio of T and B cells remained essentially constant through 9 months of age. By contrast the NZB/W female mice showed decreased numbers of total lymphocytes relative to CD-1 controls at all ages. Moreover relative to CD-1s, there was a far greater decrease in T cell numbers (7 + for NZB/W versus 2 + for CD-1) and B cell numbers failed to decrease with age. The characteristic decline in T lymphocyte numbers and relative increase in B cell numbers in NZB/W mice were corrected with cyclophosphamide and PGE1 therapy. However, there was no selective modification of T cell subsets (L3T4+ or Ly2+) with therapy. Our investigation suggests correction of the abnormal T/B cell ratio may be a useful marker of therapeutic activity in NZB/W mice.

L5 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:162567 CAPLUS
DOCUMENT NUMBER: 140:212973
TITLE: **Fluorescence** polarization assay for screening **prostaglandin** synthase inhibitors
INVENTOR(S): Ma, Y. Henry; Li, Zhuyin; Xiong, Junjie; Sabol, Jeffrey S.
PATENT ASSIGNEE(S): Aventis Pharmaceuticals Inc., USA; Hu, Linghong
SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004016223	A2	20040226	WO 2003-US25766	20030815
WO 2004016223	A3	20041021		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2495391	AA	20040226	CA 2003-2495391	20030815
US 2004082021	A1	20040429	US 2003-642468	20030815
EP 1543327	A2	20050622	EP 2003-788590	20030815
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				

PRIORITY APPLN. INFO.:		
US 2002-404008P	P	20020816
GB 2002-29244	A	20021216
WO 2003-US25766	W	20030815

AB Provided herein is a novel and useful method for evaluating the ability of compds. or agents to decrease the activity of microsomal **prostaglandin** E synthase (mPGES) or hematopoietic **prostaglandin** D synthase (hPGDS) to produce their resp. **prostaglandin** products. Such a method of the present invention comprises the steps of mixing the **prostaglandin** synthase with its substrate, a cofactor and the compound or agent so that the enzymic reaction can occur. The mixture is then incubated with a stop solution comprising an agent that prevents the spontaneous conversion of unreacted substrate into the **prostaglandin** product. This mixture is then incubated with a detection reagent that comprises the **prostaglandin** product labeled with a fluorescent **label** (i.e. a tracer), and an **antibody** having the **prostaglandin** product as an immunogen. Subsequently, the mixture and a control mixture that has been treated in the identical fashion, but lacks the compound or agent, are illuminated with plane polarized light having a wavelength at which the fluorescent **label** fluoresces. The **fluorescence** polarization of the mixture and the control mixture are measured and compared. A mixture having a polarization measurement greater than the polarization measurement of the control mixture indicates that the compound or agent decreased the activity of the **prostaglandin** synthase. The present invention is based upon the discovery that surprisingly and unexpectedly, **fluorescence** polarization can be used to identify compds. or agents that decrease the activity of a **prostaglandin** synthase, e.g., mPGES or hPGDS, to produce a **prostaglandin**, e.g., PGD2 or PGE2. An assay has been developed to measure the conversion of PGH2 to PGE2 by inducible microsomal PGE2 synthase. The assay is configured based on the

Fluorescence Polarization principle. The enzyme is incubated with PGH2, glutathione, and the compound or agent being evaluated. After a short incubation period (at least 30 s), a stop solution containing FeCl2 and citric acid is added to quench any remaining PGH2, which would otherwise undergo spontaneous conversion to PGD2 or PGE2, and thus interfere with the quantification of the enzymic conversion of PGH2 to PGE2. A detection solution containing a **fluorescence** labeled (Texas Red) tracer (PGE2) and anti-PGE2 **antibody** is then added in order to generate the specific signal that is inversely proportional to the production of PGE2. The PGE2 generated from the enzymic reaction will compete specifically for the **antibody** and release the **fluorescence** labeled tracer. Inhibition of PGE2 synthase activity will result in increased FP value. The assay described above was validated with a known inhibitor of mPGES. A **fluorescence** polarization (FP) assay to measure hPGDS activity has also been developed. The assay described above was validated using HQL 79, a known inhibitor of hPGDS.

L5 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1982:177448 CAPLUS
DOCUMENT NUMBER: 96:177448
TITLE: Binding assays
INVENTOR(S): Collins, William Patrick; Barnard, Geoffrey John Russel; Matson, Christine Mary
PATENT ASSIGNEE(S): National Research Development Corp., UK
SOURCE: Eur. Pat. Appl., 46 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 44140	A1	19820120	EP 1981-302668	19810615
R: BE, CH, DE, FR, IT, NL				
DK 8102565	A	19811214	DK 1981-2565	19810612
JP 57030952	A2	19820219	JP 1981-91456	19810613
GB 2078370	A	19820106	GB 1981-18291	19810615

PRIORITY APPLN. INFO.: GB 1980-19430 A 19800613

AB Methods for liquid differentiation immunoassay (LIDIA) of e.g. drugs and hormones are described which can be automated and which replace the commonly used solid-phase separation technique with a simple liquid-liquid partition method. This separation method can be used in conjunction with RIA and luminescence or **fluorescence** immunoassays. Thus, for the determination of estradiol in blood plasma by LIDIA, 200 μL plasma was extracted with Et2O, and to the dried extract in tubes were added estradiol-3H (as **label**) and **antibody** to estradiol. After incubation at room temperature for 30 min, UDP-glucuronyl transferase and UDP-glucuronic acid were added and incubated at room temperature for 15 min. Then scintillation fluid (PPO in PhMe-EtOH) was added, and radioactivity was determined In this procedure, free labeled hormone, after the binding reaction, reacts with enzyme in the presence of UDP-glucuronic acid to produce labeled water-soluble 17β-estradiol 3-glucuronide. When the hydrophobic scintillation fluid is added, the water-soluble conjugate is trapped in the aqueous phase, but the hydrophobic labeled hormone is released from the hormone-**antibody** complex and passes into the scintillation fluid for counting.

L5 ANSWER 6 OF 6 LIFESCI COPYRIGHT 2006 CSA on STN
ACCESSION NUMBER: 90:87762 LIFESCI
TITLE: Cyclophosphamide and 15(S)-15 methyl PGE sub(1) correct the T/B lymphocyte ratios of NZB/NZW mice.
AUTHOR: Girard, D.; Aloisi, R.M.; Bliven, M.L.; Cunningham, A.C.;

Otterness, I.G.
CORPORATE SOURCE: Cent. Res. Div., Pfizer, Inc., Groton, CT 06340, USA
SOURCE: AGENTS ACTIONS., (1990) vol. 29, no. 3-4, pp. 333-341.
DOCUMENT TYPE: Journal
FILE SEGMENT: F
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The lupus of NZB/NZW F sub(1) female mice is associated with immune complex glomerulonephritis and premature death. Cyclophosphamide and 15(S)-15 methyl PGE1 therapy halt disease progression. Fluorescein conjugated **antibodies** were utilized to **label** specific leukocytes and the subsets were quantitated using a **Fluorescence** Activated Cell Sorter. Our investigation suggests correction of the abnormal T/B cell ratio may be a useful marker of therapeutic activity in NZB/W mice.